Decompression of the Facial Nerve

A Surgical Emergency

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■ *The chance of return of normal function in cases of facial paralysis is enhanced by early surgical decompression and repair of the facial nerve. Modern precision testing is of considerable aid in prognosis as well as diagnosis. Faradic testing and electromyography can offer relatively early information as to the possibility of permanent facial deformity. Now that microscopic surgical techniques have considerably facilitated operations on the facial nerve, early operation is the treatment of choice in such cases.*

FACIAL EXPRESSIONS often play an important role in revealing character and personality. With facial paralysis, the detracting effect is relatively minor during facial repose, but when the affected person smiles, what would ordinarily be an expression of friendliness or merriment may become a twisted leer which seems to express cynicism.

The following table by Cawthorne shows some of the causes of facial palsy encountered over a period of 20 years.

Causes of Isolated Facial Palsy

	No. of Cases	*Per Cent*
Idiopathic	473	64
Injury	119	16
Geniculate	61	8
Infection	42	5
Neoplasm	28	3
Nuclear	12	1.5

The indications for surgical correction in cases of paralysis caused by injury, infection or neoplasm have been much more definite in the past than for paralysis caused by so-called idiopathic factors. This article is primarily directed toward this latter group and to support of an opinion, that earlier surgical intervention is advisable in selected cases.

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Briefly, the diagnosis of idiopathic or Bell's palsy should be restricted to cases in which the facial palsy is the only clinical symptom with no obvious cause to be found on examination.

The facial nerve lies in a narrow, tortuous tube encased in the temporal bone. Any reactive swelling of the nerve or its sheath may squeeze the blood out of the vessels which nourish the nerve and thus deprive it of the oxygen vital to its function. The chain of events in Bell's palsy is as follows: Primary ischemia caused by vasospasm results in venous stasis and edema of the facial nerve within the facial canal. The edematous nerve in turn causes secondary ischemia, with further impairment of circulation. Oxygen deprivation is the result of the vicious cycle thus established.

Inasmuch as many patients with Bell's palsy recover completely without medical or surgical intervention, neurologists in most centers have for many years advocated watchful expectancy. However, the patient who does not obtain satisfactory return of function, after months of agonizing suspense, receives little comfort in knowing that in most similar cases the patient fared better.

Refinement of diagnostic methods and concepts in recent years have made possible more precise prognostications and therapeutic indications. Electrodiagnosis has become increasingly useful in determining the extent and severity of the under-

lying physiological disturbance. Absence of faradic reaction (interrupted current) suggests early Wallerian degeneration. If there is no galvanic reaction (continuous current) muscle atrophy is indicated. When no contraction is present with either current it is because the reaction of degeneration has occurred. Electromyography provides further confirmation of reaction of degeneration if fibrillations occur on the tracings.

Cawthorne[2] reported that patients with incomplete palsy, as determined by clinical and electrical findings, have a relatively good prognosis without special therapy. Ninety-five per cent of these patients recover spontaneously. However, those who have complete palsy, with proven reaction of degeneration, have a much less favorable prognosis without surgical therapy. Only 45 per cent of such patients recover completely. Thus, with nerve conduction tests it is possible to predict which patients may recover quickly and completely and which slowly and not fully.

Electrodiagnostic studies are indicated at twice weekly intervals following the onset of the facial paralysis. Should reaction of degeneration occur, immediate surgical decompression of the seventh nerve is indicated. Kettle[3] and others have demonstrated that the prognosis for return of function is greatly enhanced with such a course of action.

The advent of microscopic surgical techniques has greatly increased the confidence and competence of otologists working in the region of the facial nerve. Exposure and decompression of the nerve is no longer a formidable task. Some difference in opinion exists as to which surgical approach, endaural or post-auricular, is most satisfactory. The endaural approach offers the best view of the nerve where it first makes its descent in the vertical portion of the canal. On the other hand the post-auricular approach is advantageous because the stylomastoid foramen can be enlarged much more easily and this is the most important part of the procedure of decompression. Inasmuch as incisions in and about the ear heal so nicely, I make use of a combination of both incisions in order to obtain optimum working conditions in all areas. Ten power microscopy is used for examining the operative field during the initial exposure of the nerve with a 4 mm motor-driven burr. In the process of bone removal there is a stage just before actual exposure of the nerve when a reddish streak appears that indicates the proximity of the nerve sheath. That is the point at which the bony covering of the canal has been reduced to a fraction of a millimeter in thickness and is sufficiently transluscent to make the underlying vascular structure of the nerve sheath visible. A smaller burr is then used to lathe a sulcus parallel to the nerve on either side of its entire vertical portion, almost exposing the sheath. This hollow grinding technique makes it much easier to make gentle use of a small stapes curette to remove the final layer of thin bony plate overlying the sheath. Three-quarters of the nerve circumference is thus exposed. The compressed blood vessels thereupon fill with blood and the nerve bulges to varying degrees. Care must be taken to carry the decompression well into the stylomastoid foramen. I do not slit the sheath and I cannot see the rationale of doing so if the nerve is otherwise adequately decompressed. The incisions are closed without drainage after the nerve is covered with Gelfoam pledgets and a fascia pedicle is dropped into the inferior portion of the mastoid cavity.

Reports of Cases

CASE 1. A 16-year-old boy, examined on referral 15 November 1963, had awakened nine weeks previously with pain at the base of the skull. He noted that he could not move the left side of his face. The pain disappeared shortly but the sense of taste was impaired for about a week. A neurosurgeon attending the patient ordered electrodiagnostic testing at intervals by a qualified physiatrist. These examinations indicated gradual deterioration of the facial nerve to a stage of complete reaction of degeneration.

Surgical decompression of the facial nerve was performed on 19 November, approximately 10 weeks after onset of the paralysis and one week after complete reaction of degeneration was first noted. The nerve sheath was well exposed from the level of the horizontal semicircular canal to the digastric groove. The sheath was bright red in color but bulged only slightly when freed from its bony prison. No attempt was made to achieve further decompression by slitting the epineurium. Gelfoam pledgets were used to cover the nerve and a fascia pedicle was draped into the mastoid cavity. The skin incisions were completely closed and the patient was sent home two days after operation. Electrical stimulation was begun again 10 days after operation and encouraging responses were noted a few days later and within a month voluntary movements of the affected side were

easily detectable. The attending neurosurgeon prescribed the use of a small Waters electrical stimulator for home use, and when the patient was seen two months later most of the facial function had returned except that the patient could not wrinkle the left side of his forehead and had to concentrate to completely close his left eyelid. The latter function thereafter gradually improved but the forehead musculature remained immobile.

CASE 2. A 42-year-old woman who was examined on referral 15 January 1965 had wakened approximately seven weeks previously to find that she had difficulty in moving the left side of her face and could not close her left eye. She immediately saw an internist who prescribed vitamin B-12 and referred her to a physiotherapist for treatments. As the treatments did not help, the patient was referred to me for opinion and therapy as indicated. Electrodiagnostic tests performed on 18 January disclosed complete reaction of degeneration and fibrillation to electromyography. Decompression of the facial nerve was carried out the following day, almost two months after the onset of symptoms. Excellent exposure of the nerve was accomplished without difficulty. The sheath was quite red, moderately swollen and trabeculated, with small vessels on the surface. No attempt was made to decompress the nerve further by slitting the sheath. Gelfoam strips were used to cover the nerve and the incisions were completely closed. The patient was sent home after two uneventful postoperative days. Eight days after operation, barely perceptible voluntary twitches could be noted in the corner of her mouth. Facial function then steadily improved to the extent that, less than two months after operation, all natural facial creases were present. Home use ot a small Waters electrical stimulator was prescribed for supplementary exercise several times daily.

Discussion

Ordinarily, in cases in which complete reaction of degeneration occurs, beginning signs of recovery are not expected for seven to nine months. In the cases here reported, recovery began much sooner—three weeks after operation in one instance and only one week afterward in the other.

Certainly in any case of idiopathic facial palsy medical care should be instituted as soon after onset as possible, with use of nicotinic acid and corticosteroids to bring about vasodilation and reduce secondary edema. Some investigators advocate supplementing such a program with the use of stellate ganglion block, a procedure with which I have had no experience. Clinical observations should be correlated with serial nerve conduction tests and electromyography. Should deterioration of function continue to the extent of complete paralysis and reaction of degeneration, surgical decompression should be performed without delay.

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